1535 WHICH MATURE BABIES NEED TO BE OBSERVED IN THE NURSERY: ANALYSIS OF THE SURFACE TENSION (ST) OF AMNIOTIC FLUID (AF) LIPID EXTRACT (LE). Chandra M. Tiwary, James B. Haddock, Richard D. Landes, and Doris Burgess (spons. Andrew W. Margileth). Dept. of Peds., Walter Reed Army Med. Ctr., Wash., D.C., and Uniformed Services Univ. of the Health Sciences, Bethesda, Maryland.

We reported (Ped. Res. 1981:15:1452A) that the mothers whose AF LE showed reduced ST lowering property delivered babies who developed complications in the neonatal period. This study included babies of all weights. To exclude the impact of premies we examined the predictive value of ST lowering property of the AF LE for newborns weighing ≥2500gm. The ST was measured on 64 AF LE by the standard method. The ST sum was calculated by adding the volume and the ST (both are the minimum volume (ul) of the AF LE required to maximally lower the ST (dynes/cm)).

In 28 babies (♂15,♀13), the ST sum was≤40; 22 were normal and 6 (♂5,♀1) showed complications: meconium staining-3, ABO incompatibility-2, hyperbilirubinemia requiring phototherapy-2, and Down's syndrome-1. In 36 babies (♂17,♀19), the ST sum was >40; 19 were normal and 17 (♂6,♀11) showed complications: Rh and other isoimmune hemolytic diseases requiring exchange transfusion-4, hyperbilirubinemia requiring phototherapy-2, ABO incompatibility-2, polycythemia requiring partial exchange transfusion-1, hypoglycemia-4, possible sepsis-1, and meconium staining-3. The mothers of only 5 babies showed a prenatal condition suggesting a need for the baby's observation. Conclusion: ST sum value is a nonspecific indicator of a baby's health. A high value suggests a need for observation of a ≥2500gm baby; a low value suggests an absence of complications subsequent to delivery.

†1536 Effect of Prenatal Glucocorticoid on Fetal Rat Lung Prostaglandin Synthesis. Michael Y. Tsai, Mark W. Josephson, Bill Handschin, David M. Brown, Department of Lab Med and Pathology,Univ of Minnesota, Minneapolis, 55455. Prenatal Glucocorticoid therapy is increasingly being used for accelerating fetal lung maturation. Glucocorticoids, however, are also known to inhibit phospholipase A_2 and thus the synthesis of prostaglandins(PG). In perinatal rat lung, the major PG is prostacyclin(PGI_2), a potent vaso- and bronchodilator important in lung function. To determine the effect of gluococorticoid therapy on fetal lung PGI_2 synthesis, we measured 6-keto-$PGF_{1\alpha}$ (the stable breakdown product of PGI_2) levels by RIA. Pregnant rats received 4 doses of dexamethasone(DEX)(0.4mg/kg) at 12hr intervals prior to sacrifice. Table 1 shows the 6-keto-$PGF_{1\alpha}$ levels of fetal lungs from DEX-treated and control mothers (mean ± SEM, 4 fetuses from each of 6 litters for each group).

Table 1 6-keto-$PGF_{1\alpha}$ (pg/mg protein)

DEX Treatment	21 Days Gestation	p	22 Days Gestation	p
Control	292 ± 49		256 ± 36	
0.4mg/kg	443 ± 49	0.05	443 ± 28	0.002

DEX treatment significantly increased 6-keto-$PGF_{1\alpha}$ levels. There were no significant differences between male and female fetuses with or without DEX treatment. GC/MS studies confirmed results obtained by RIA. These results suggest that prenatal DEX enhances endogenous levels of 6-keto-$PGF_{1\alpha}$ in fetal lung. Since PGI_2 may be important in perinatal lung maturation and function, the effectiveness of glucocorticoid therapy for accelerating functional lung maturity may be partly due to the stimulation of PGI_2 synthesis.

●1537 ELEVATED CALCITONIN (CT) IN BIRTH ASPHYXIA AND PREMATURITY: ROLE IN THE PATHOGENESIS OF EARLY NEONATAL HYPOCALCEMIA (HC) P. Venkataraman, R.C. Tsang, I. Chen, M. Sperling, Dept. Pediatr., Univ. of Cincinnati

Although CT is stress responsive the role of CT in pathogenesis of early neonatal HC is unknown. We studied the thesis that CT, gastrin, glucagon 1) are higher in cord than mother; 2) rise postnatally; 3) correlate inversely with gestation; 4) are higher in birth asphyxia; and 5) elevated CT results in HC; 6) gastrin and glucagon are CT secretagogues. We studied 64 mother-infant pairs, gestation 25-42 wks, Apgar 1' 6.2±2.7, 5' 7.6± 2.2. Cord Ca, Mg, P (mg/dl), CT, gastrin and glucagon (pg/ml) were mostly higher than maternal, 10.15±(SEM) 0.18 vs 8.8±0.16 ($p< 0.005$); 1.95±0.06 vs 1.8±0.06 ($p<0.05$); 5.8±0.25 vs 3.4±0.13 ($p<.005$); 81±17 vs 49±11 ($p<0.05$); 133±20 vs 123±15 (n.s.); 120±9 vs 78±7 pg/ml ($p<0.005$) respectively. In neonates at 24 h CT, gastrin and glucagon rose to 254±29 ($p< 0.005$); 172±28 (n.s.); 216±17 pg/ml ($p<0.005$). Serum Ca fell to 8.7±0.2, 8.7±0.3 mg/dl at 24, 48 h, ($p<0.005$). Term cord CT correlated with 1' Apgar, $r=-0.4$ ($p<0.05$), at 5', $r=-0.8$ ($p< 0.0001$). 24 h serum CT correlated with 24 h serum Ca, $r=-0.7$ ($p<0.0003$) and 48 h Ca $r=-0.93$ ($p< 0.0003$). Cord CT was higher <32 wks vs term 146±45 vs 61±18 pg/ml ($p<0.05$) and higher with Apgar <6 vs >7 at 1' and 5', 118± 37 vs 56±18 and 266±72 vs 49±9 pg/ml resp ($p<0.05$). Neither serum gastrin nor glucagon correlated with CT. Thus, 1) cord CT and glucagon are elevated; 2) CT and glucagon rise postnatally; 3) cord CT is higher in preterm and asphyxia; 4) high serum CT correlates with low serum Ca. We speculate that elevated serum CT may result in HC in preterm and birth asphyxiated infants.

●1538 FUROSEMIDE EFFECTS ON NEWBORN RENAL & BONE CALCIUM METABOLISM, Zhi-Ping Guan, Winston Koo, Jerry Schutzman, Vicky Neumann, & Reginald C. Tsang, University of Cincinnati College of Medicine.

Furosemide diuretics are commonly used in neonatal intensive care. Recent anecdotal reports have appeared of preterm infants who develop renal calcification & osteopenia on chronic high dose furosemide therapy. The mechanisms for development of these possible complications in infancy is unclear. We hypothesize that furosemide diuretics result directly in hypercalciuria, nephrocalcinosis, secondary hyperparathyroidism & decreased bone mineral content. Newborn rats were randomized from day four into control & treated groups for a 28 day study. Grp. 1, placebo; Grp. 2, daily 5 mg/kg of furosemide; Grp 3, 15 mg/kg of furosemide. By analysis of variance, urinary calcium increased from 7.81 to 11.25 to 20.35 mg/dl for the three respective groups ($p < .05$). Urinary Mg also increased from 13.1 to 14.1 to 19.3 mg/dl. Urinary P did not increase. Renal Ca ash content of treatment grps. were significantly increased (6 of 25 & 6 of 26) beyond control 95% limit. Chi-square $p<.05$. Bone weight of tibias was decreased from .21 to .17, .16 gs. ($p<.01$), as was ash weight .13, .11, .10 gs. ($p<.05$), in association with decreases in body weight of 68, 62, 57 gs. Bone Ca & body weight were correlated ($p .01$). Serum Ca, Mg, P, & parathyroid hormone concentrations (mid molecule 44-68 radioimmunoassay, rat standard, CV 9%), were not different among grps. Thus, furosemide in newborn rats results in increased urinary Ca, increased renal Ca content, decreased bone mineral, decreased body weight & no changes in serum Ca, Mg, P or parathyroid hormone. We speculate that the effect of furosemide therapy in the newborn on Ca metabolism is directly related to increased Ca loss in the urine.

1539 LONG TERM FOLLOW-UP IN ≤1500 GM. BIRTHWEIGHT (VLBW). J. G. Urrutia, T. Mathew, E. Brookfield, M. Satish, S. McQuiston, J. Butterfield, S. ElShafie (Spon. by M. G. Robinson), Medical College of Ohio, The Toledo Hospital, Dept. of Ped., Toledo, Ohio.

376 VLBW neonates were admitted to Regional Perinatal Center, between July 1979 to December 1981. 281 (75%) survived, 127 (45%) were followed up for up to 18 months corrected age and 3 years of age. The overall neonatal mortality was 25%; mean gestational age, 29.8 ± 2.35 wk.; mean birthweight, 1113 ± 230 gm.; SGA, 26%; Apgar ≤3 at 1 min., 35%; ≤5 at 5 min., 21%; outborn 35%, ventilated 78%. CT scan/ultrasound was done on 77 (61%), of which 38 (49%) had paraventricular-intraventricular hemorrhage. Neurologic examination, Bayley Scales of Infant Development, McCarthy Scales of Children's Abilities were done. Cerebral palsy or developmental delay (MDI more than 2 standard deviations below the mean), visual deficits were considered severe handicaps. Mean Bayley Score 70-84 were considered suspect.

BIRTHWEIGHT (g)	NO HANDICAP	SUSPECT	SEVERE HANDICAP
500 - 750	5	1	3
751 - 1000	20	9	5
1001 - 1250	26	9	1
1251 - 1500	25	14	9
TOTAL	76 (60%)	33 (26%)	18 (14%)

Our data confirms optimistic results of modern perinatal care. Additional work is needed to further reduce incidence of handicap.

1540 LONG TERM FOLLOW-UP IN VERY LOW BIRTHWEIGHT (VLBW) NEONATES WITH PARAVENTRICULAR INTRAVENTRICULAR HEMORRHAGE. J. G. Urrutia, T. Mathew, E. Brookfield, M. Satish, S. McQuiston, J. Butterfield, S. ElShafie. (Spon. by M. G. Robinson) Medical College of Ohio, The Toledo Hospital, Dept. of Ped., Toledo, Ohio.

78 VLBW admitted to the Neonatal Intensive Care Unit, in the Regional Perinatal Center, during the period July 1979 to December 1981 had CT brain scan and/or ultrasound. They were followed at 18-24 months corrected age and neurologic examination and Bayley Scales of Infant Development were performed. Their gestation (wks.) and birthweight (kg.) in the control group were G.A. 29.3±1.9 and birthweight 1.050±0.2 and paraventricular-intraventricular group were G.A. 29.2±2.3 and birthweight 1052±.3. Degree of hemorrhage was graded according to Papile.

	Normal	G I	G II	G III	G IV
(N)	40	8	8	14	8
Major Handicap	4(10%)	1(13%)	0	5(36%)	7(88%)
MDI Mean±S.D.	88±17	87±11	91.5±24	68.4±16	59±17
PDI Mean±S.D.	85±12	86±8	87.1±10	77±14	50±8.5

Progressive significant motor and mental handicaps were found with Grade III and IV hemorrhage.